American Petroleum Institute

1220 L Street, Northwest
Washington, DC 20005-4070
Tel: 202-682-8344
Fax: 202-682-8270
E-mail: twerdokl@api.org

Lorraine E. Twerdok, Ph.D., DABT
Manager, Health Sciences

April 7, 2000

Carol Browner, Administrator
US Environmental Protection Agency
P.O. Box 1473
Merrifield, VA 22116
Attention: Chemical Right-to-Know

RE: HPV Test Plan and Robust Summaries Submission for Petroleum Coke
HPV Challenge Program, AR-201
HPV Consortium # 1100997

Dear Ms. Browner:

The American Petroleum Institute, on behalf of the Petroleum HPV Testing Group, is pleased to submit the Petroleum Coke Test Plan and Robust Summaries. Our consortium has chosen not to use the HPV Tracker system for submission of our test plans due to the complexity of petroleum substance categories and subsequent test plans. We are therefore submitting the test plan, as well as robust summaries directly to EPA to make available for public comment.

One hard copy and one electronic in .PDF format are contained in this HPV Challenge Program, AR-201 submission. This submission is also being sent, via email, to the EPA HPV robust summary submission address and Charles Auer.

Please feel free to contact me (202-682-8344; twerdokl@api.org) or Tom Gray (202-682-8480; grayt@api.org) with any comments or questions you may have concerning this submission.

Sincerely,

Cc: C. Auer (via email)
HPV Robust Summary Emailbox
T. Gray

PETROLEUM COKE TEST PLAN

Submitted to the US EPA

by

The American Petroleum Institute
Petroleum HPV Testing Group

Consortium Registration # 1100997

PETROLEUM COKE TEST PLAN

Table of Contents

PETROLEUM COKE TEST PLAN

PLAIN LANGUAGE SUMMARY

In petroleum refineries, useful products like gasoline, jet fuel, diesel fuel, motor oils and waxes are separated from crude petroleum, leaving a heavy tar-like residue. More product can be made from this heavy residue by processing it at high temperatures and pressure to crack large molecules into smaller molecules. This process, called coking, leaves behind a hard, coal-like substance called petroleum coke. It consists mostly of carbon with smaller amounts of hydrocarbons (oil) and sulfur, and trace amounts of metals.

This category includes petroleum coke and calcined coke. Petroleum coke, also called green coke, is used primarily as an industrial fuel. Green coke can be further processed at very high temperatures to make calcined coke. This calcining process removes nearly all of the residual oil so that calcined coke consists mostly of pure carbon, with trace amounts of sulfur and metals. The lower oil content makes calcined coke a dustier material than green coke. Calcined coke is used to make synthetic graphite and electrodes for smelting furnaces.

Since coke is made up largely of carbon, and carbon is an inert substance, the potential for coke to cause acute toxicity (harm from exposure to large quantities) is very low. For that reason, health studies conducted by the petroleum industry have looked for possible chronic toxicity, or harm from repeated exposures to the dust over a long period of time. Most of these studies used green coke because of its higher oil content; calcined coke would be expected to have a lower hazard since it is almost entirely carbon.

Exposure to high concentrations of any inert dust can, over time, cause mild to moderate inflammation of the lung, and this was confirmed in laboratory animal studies using high concentrations of green coke dust. These studies, done in rats and monkeys, lasted for two years and showed inflammatory changes in the lungs and nose from the build-up of dust particles. Slight scarring of the lung tissue was seen in rats, but not monkeys. Another important finding was that repeated daily exposures did not cause cancer or other harmful effects on any of the body's organ systems. These studies used concentrations of green coke that were six times higher than the safe level set by OSHA for workplace exposures.

Studies in mice in which green coke dust was applied three times per week to the skin for two years did not produce skin cancer or other effects on the skin. Green coke was also tested for its ability to alter DNA (cause mutations) in a variety of cell culture and animal studies, and those studies were negative. The only endpoints which have not been studied are reproductive effects, and effects on the developing fetus. Those studies will be initiated this year using green coke.

Because petroleum coke is a solid, fairly inert material there has not been much concern about its environmental effects. The typical battery of tests used to measure a chemical's impact on the environment, such as breakdown by sunlight, stability in water, breakdown in the soil and volatility, can not be measured for petroleum coke. In addition, the lack of significant adverse effects in animal studies suggests that the residual oil associated with petroleum coke either does not come off, or is present at amounts too low to cause harmful effects. To evaluate environment effects, samples of green coke will be tested in aquatic organisms, plants and earthworms.

In summary, health studies done on green coke have confirmed its low toxicity. It is reasonable to assume that these findings also apply to calcined coke because it is almost pure carbon. Testing for reproductive effects and possible harm to the fetus are planned for green coke. Green coke will also be studied for possible harmful effects in aquatic organisms, plants and earthworms.

DESCRIPTION OF PETROLEUM COKE

Petroleum coke is a black solid produced through the thermal decomposition of heavy petroleum process streams and residues. The feedstocks undergo cracking and carbonization to a product with a high carbon to hydrogen ratio, which may be granular or needle-like in appearance. Petroleum cokes can be categorized generally as either green or calcined coke. The initial product of the coking process, green coke, is used as a solid fuel. Further processing of green coke at higher temperatures and pressures result in calcined coke which is used in the manufacture of electrodes, in smelting applications, for graphite electrode production, or for minor applications such as carbonization of steel.

Coking Processes and Terminology

Green coke can be produced by one of three processes: delayed, fluid or flexicoking.

Delayed process coke is produced by a semi-continuous batch process and accounts for more than 95% of total US coke production (1). Green coke comes from the coke drum in large pieces and is milled to a smaller size before subsequent use as a fuel or as feedstock for calcining. Green coke can contain as much as 15% residual hydrocarbon, which gives it a characteristic hydrocarbon smell.

Fluid coke is produced by a continuous fluidized bed process. Fluid coke typically contains less residual hydrocarbon than delayed process green coke but more than calcined coke, and occurs as spherical grains less than 1 cm in diameter.

Flexicoke is produced by a variant of the fluidized bed process in which most of the coke is converted to a low Btu fuel gas for use in the refinery in which it was produced. Solid flexicoke has a smaller particle size than fluid coke and is dustier due to its lower residual hydrocarbon content.

Calcined coke is produced from delayed process green coke by a process of further heating at temperatures up to 1200^{o}C. The product of calcining removes virtually all of the residual hydrocarbon including PAHs and the result is a dustier material.

Calcined coke is characterized as either anode-grade coke or graphite needle-grade coke depending upon its physical and chemical characteristics with needle-grade coke having a higher purity than anode-grade coke which is used in electric furnaces in aluminum and steel smelting.

Depending on its physical form, coke may also be classified as shot, sponge or needle coke. Shot coke occurs as hard spheres and is produced from high asphaltene precursors. Needle coke appears as silver-gray crystalline needles and is derived from feedstocks with high aromatic hydrocarbon content. Sponge coke is dull black with a macroscopically amorphous appearance but is a mixture of shot and needle coke structures.

Green coke and calcined coke are covered by definitions of petroleum coke in the EINECS system. They are: Petroleum coke, and Coke (petroleum) calcined. The definitions of these categories are listed in Appendix 1.

Analytical Characterization

Petroleum coke is characterized by its chemical composition and physical characteristics. The chemical composition of petroleum coke is dependent upon the composition of the feedstocks that are used in the coking process, which in turn are dependent upon the composition of the crude oil from which they are derived. The metals and sulfur composition of calcined coke is directly dependent upon the composition of the green coke from which it was produced.

The physical characteristics of petroleum coke are important in determining the suitability of a coke sample for a specific use. These are typically the real and bulk densities and, in the case of anode and needle grade coke, the resistivity and coefficient of thermal expansion. Fuel grade coke may also be characterized by its fuel value (btu/lb).

Typical parameters measured to define the chemical composition of petroleum coke are:
Weight % ash, weight % sulfur, weight % residual hydrocarbon, ppm nickel, and ppm vanadium. Residual hydrocarbon includes organic matter ranging from 6-carbon compounds to 7-ring polycyclic aromatic hydrocarbons (PAHs). Because of the lower temperature used in its production, green or fuel-grade coke contains higher levels of residual hydrocarbon than other grades of coke. The calcining process removes essentially all of the residual hydrocarbon (< 0.5 %).

The following table illustrates the difference between fuel grade green coke and coke intended for aluminum anode grade before and after calcining. These two grades (fuel and calcined) are representative of the two extremes of petroleum coke composition.

Table 1[1]

Properties[2]	Fuel-Grade Green	Anode-grade calcined
Sulfur (wt%)	2.5 – 5.5	1.7 – 3.0
Ash (wt%)	0.1 – 0.3	0.1 – 0.3
Nickel (ppm)	N.D.[3]	165 – 350
Vanadium (ppm)	200 – 400	120 – 350
Residual hydrocarbon (wt%)	9 – 12	<0.25
Bulk density (g/cm3)	N.D.	0.80
Real density (g/cm3)	N.D.	2.06

[1] From Lee et al., 1997
[2] The above values are given for illustration and may vary depending upon the feedstock or crude oil of origin
[3] Not determined

The residual hydrocarbon portion of green coke has been shown to contain PAHs. The PAH content roughly parallels the residual hydrocarbon content of the different grades and processing temperatures of coke. Delayed process green coke, with the highest residual hydrocarbon content was reported to contain higher levels of PAHs than fluid process coke (2). There has been no correlation demonstrated between PAH content and animal or genetic toxicity (see health effects data below).

TEST MATERIAL JUSTIFICATION

This category contains petroleum (green) coke and calcined coke. Both are composed primarily of elemental carbon. The principal attribute that distinguishes green coke from calcined coke is the concentration of residual hydrocarbon. The extremes in composition range from green coke, with relatively high residual hydrocarbon content, to calcined coke with less hydrocarbon and higher elemental carbon. Calcined cokes always contain significantly less residual hydrocarbon than green coke and generally less sulfur. The metal content of calcined coke may be higher than in green coke since the loss of residual hydrocarbon increases the relative content of the metals.

Because elemental carbon is known to be biologically inert, and the residual hydrocarbon content is highest in green coke, health and environmental effects testing will be conducted using green coke. If health or environmental impacts are possible, testing green coke will provide the greatest likelihood of detecting these effects. To date, no adverse effects have been reported which can be attributed to the residual hydrocarbon portion of petroleum coke.

EVALUATION OF EXISTING HEALTH EFFECTS DATA AND PROPOSED TESTING

As described above, the petroleum coke category includes green coke and calcined coke. There have been no human studies of the possible health effects of these materials, but there have been epidemiology studies at manufacturing plants where petroleum coke was in use (3). These studies focused on the effects of dust on respiratory function. Employees completed a medical questionnaire and underwent pulmonary function

tests and chest X-rays. The medical evaluations demonstrated decrements in pulmonary function typical of any dusty work environment, and related decreases in pulmonary function to the amount of coke dust exposure. Chest X-rays did not show any indication of pneumoconiosis or chronic lung inflammation.

The majority of the animal toxicity studies available for evaluation has been on green coke (either delayed or fluid process) which has a higher residual hydrocarbon content than calcined coke. In one repeat dose study calcined coke was tested for comparison. An evaluation of the existing studies as well as plans for further studies is summarized below. A matrix (Table 2) summarizing this information can be found at the end of the test plan.

Acute Toxicity

There are no acute oral or dermal toxicity studies available on either green or calcined coke. Because petroleum coke materials are comprised largely of elemental carbon, which is biologically inert, they are considered to have a low degree of acute toxicity. Limited support for this comes from workplace experience and the results of oral and dermal LD 50 studies of carbon black, another carbonaceous material containing organic hydrocarbons albeit in much lower concentrations. LD 50 studies of carbon black resulted in values of >15.4 g/kg and > 3 g/kg respectively for the oral and dermal routes (4). Similarly, feeding studies of thermal black, which contained up to 14 % extractable hydrocarbon, noted no grossly observable effects after up to 72 weeks of exposure to 10% test material in the diet (5).

Repeated dose subacute inhalation studies have been conducted to compare lung responses to green and calcined coke. In these studies, rats were exposed by nose-only inhalation for 5 days to either calcined coke (45 mg/m3) or green coke (58 mg/m3) (6). A silicon dioxide exposure group was also used as a benchmark to judge the potential for the coke samples to cause lung fibrosis. Lung response was evaluated by analysis of lung fluids at 7, 28 and 63 days after exposure. Both green and calcined coke caused slight inflammatory responses in the lung, with the response to green coke being slightly higher. There was no indication of a fibrogenic response from exposure to either the green or calcined coke.

Additional support for the low inhalation toxicity of coke is provided by two-year inhalation studies of green coke in rats and primates in which no signs of systemic toxicity were observed (7,8). The whole body nature of the rat exposures would have resulted in oral ingestion of some test material as a result of grooming. A similar lack of systemic toxicity was noted in a two-year dermal bioassay of green coke suspended in mineral oil (9). The lack of effect from chronic exposures supports the position that acute toxicity of green or calcined coke would be low.

Summary: There are no acute oral or dermal toxicity or irritation studies available on petroleum coke, however, workplace experience, studies of similar materials, and chemical composition suggest a low degree of acute toxicity for these materials. Subacute inhalation studies of both green and calcined coke demonstrated a low degree of toxicity. No acute toxicity studies are planned for this category.

Repeat Dose Toxicity

Repeated dose inhalation toxicity studies of green coke (delayed process) have been conducted in both rats and monkeys. Both studies were conducted at concentrations of 10 and 30 mg/m3 of green coke, which had been micronized into fine particles to aid in aerosol generation (7,8). Both species were exposed for two years. Blood chemistry, clinical chemistry, comprehensive eye examinations and thorough gross and microscopic examinations were conducted at 3-month intervals. The only treatment related effect reported was inflammatory changes in the lungs, caused by the accumulation of fine dust particles. The lung inflammation in rats was greater than in monkeys, in some cases leading to a slight scarring of the lung tissue as a result of chronic inflammation. This is considered a typical response of the lung to high concentrations of dust. Eye evaluations conducted in both studies did not reveal any adverse effects from the exposures. Petroleum coke was not found to be carcinogenic by the inhalation route in either the rat or monkey study.

Repeated dose dermal toxicity studies have been conducted on green coke (both delayed and fluid process). Each of the petroleum coke samples was micronized and suspended in mineral oil, and applied to mouse skin three times per week for two years (9). The only effect observed in the mice was a thickening of the skin in the area of treatment. Neither coke sample caused skin cancer in this study.

Summary: The repeated dose inhalation and dermal toxicity studies indicating low toxicity for green coke are judged adequate for assessing the hazard from inhalation and dermal exposure to petroleum coke. No further testing is planned.

In Vitro Genetic Toxicity

Green coke (both delayed and fluid process) have been evaluated for bacterial mutagenicity in the Ames test using Salmonella typhimurium strains TA 1535, 1537, 1538, 98 and 100, with and without metabolic activation (10,11). None of the coke samples produced a positive response in any of the tester strains.

The delayed and fluid process coke samples were also evaluated in a mammalian cell mutagenicity test using the L5178Y mouse lymphoma cell line (10,11). The tests were conducted with and without metabolizing enzymes in the assay system. Neither coke sample was mutagenic.

Summary: In vitro genetic toxicity testing of green coke has been conducted in both bacterial and mammalian cells with no indication of genetic toxicity. Since green coke has a higher residual hydrocarbon content, these studies are considered adequate to assess the potential for genetic toxicity of calcined coke. No further in vitro mutagenicity testing is planned for this category.

In Vivo Genetic Toxicity

Green coke (both delayed and fluid process) has been evaluated for its ability to produce chromosome aberrations in a bone marrow cytogenetic assay (10,11). Rats were exposed via inhalation to concentrations of 10 and 40 mg/m3 coke dust, and at the end of the study the bone marrow analyzed for chromosomal changes. No changes were seen in animals exposed to the fluid process coke for 20 days. In animals exposed to the delayed process coke for 5 days, an increase in the number of chromosome abnormalities was seen at the high dose only. The laboratory later determined that the slides had been misread, and that the results for the delayed process coke should be considered inconclusive.

Because of uncertainty about the reported positive results in the delayed process coke study, additional bone marrow samples were obtained from animals used in the two-year study described above under "repeated dose toxicity" (7). Analysis was conducted on animals after exposure for 5 days, 12 months and 22 months to concentrations of 10 and 30 mg/m3 coke dust. No abnormal chromosome changes were seen in any of those animals.

Summary: The potential for green coke to cause chromosome abnormalities has been evaluated in two separate inhalation studies with no indication of genetic toxicity. Since the residual hydrocarbon content of green coke is higher than that of calcined coke, it is considered unlikely that calcined coke would cause genetic toxicity in similarly conducted studies. No further testing is planned.

Reproductive And Developmental Toxicity

No studies have been conducted on green or calcined coke to determine the potential for causing adverse reproductive effects or adverse effects on the developing fetus. Repeated dose studies conducted on green coke (described above) did not identify any harmful effects on the reproductive organs.

Summary: Because green coke has a higher residual hydrocarbon content, it will be used in a reproductive and developmental toxicity screening test, conducted by inhalation (OECD 421).

EVALUATION OF EXISTING PHYSICOCHEMICAL AND ENVIRONMENTAL FATE DATA

The physicochemical endpoints in the HPV chemicals program include:

- Melting Point
- Boiling Point
- Vapor Pressure
- Octanol/water Partition Coefficient
- Water Solubility

The environmental fate and effects endpoints include:

- Photodegradation
- Stability in Water (Hydrolysis)
- Transport and Distribution (Fugacity)
- Biodegradation
- Acute Toxicity to Fish
- Acute Toxicity to Aquatic Invertebrates
- Toxicity to Algae (Growth Inhibition)

Developing meaningful physicochemical and environmental fate and effects information/data for materials in the Petroleum Coke category will not be possible for certain endpoints because of their chemical structure and physical nature. Materials in this category are amorphous solids, composed primarily of carbon and are either not amenable to standard testing guidelines or the endpoints of interest are not relevant in view of their chemical structure. Petroleum Coke is also not subject to structure based modeling because it does not have a single, unique chemical structure.

Both green and calcined cokes are relatively inert and would not be expected to interact with the environment in an adverse manner. This is perhaps why little environmental information has been developed for these materials. However, with the exception of the aquatic toxicity endpoints, relevant environmental information will be summarized in technical discussions for the endpoints listed above. Limited aquatic toxicity testing will be conducted to assess the potential toxicity of these materials.

Petroleum Coke is used as a soil amendment to improve the insulative capacity of soils beneath power stations. The fate and effects endpoints in the HPV chemicals program do not include soil toxicity tests. Nevertheless, this testing plan will include selected terrestrial toxicity studies to develop data for this purpose.

Physicochemical Data

Physicochemical data for the Petroleum Coke category that can be used in the HPV chemicals program were not found. There are estimation structure-activity relationships for the physicochemical endpoints in the computer program EPIWIN (12) (Estimation Program Interface for Windows) and EPA has suggested that subroutines in this program would be acceptable to develop data for these endpoints (13). However, this program functions using specific structure based rules that cannot be applied to structures representative of the Petroleum Coke category. There is more information on the use of EPIWIN for the HPV chemical program in the EPA document titled, *The Use of Structure-Activity Relationships (SAR) in the High Production Volume Chemicals Challenge Program*.

Developing measured data for a material representative of the Petroleum Coke category is limited by the fact that these materials are solids and water insoluble under relevant environmental conditions. As an alternative to not being able to model or develop measured data, technical discussions will be developed that characterize selected physicochemical properties of materials in the Petroleum Coke category.

Summary: Testing and computer modeling of physicochemical endpoints will not be conducted for materials in the Petroleum Coke category because their structure precludes developing data in either of these manners. Instead, technical discussions on each property will be prepared and added to IUCLID, which is the electronic database that will contain data for the HPV chemicals program.

Environmental Fate Data

Environmental fate data for the Petroleum Coke category that can be used in the HPV chemicals program were not found. The following describes the fate endpoints and the type of information that will be developed.

Photodegradation: Direct photochemical degradation occurs through the absorbance of solar radiation by a chemical substance. If the absorbed energy is high enough the resultant excited state of the chemical may undergo a transformation.

Photodegradation can be measured (14) (EPA identifies OECD test guideline 113 as a test method) or estimated (13). An estimation method accepted by the EPA includes the calculation of atmospheric oxidation potential (AOP). However, only chemicals that have a potential to enter a vapor phase will be available for atmospheric oxidation reactions with photochemical generated hydroxyl radicals. Because Petroleum Coke is a solid for which a representative molecule that would be amenable to modeling does not exist, the accepted procedures used to assess photodegradation are not appropriate. Therefore, to satisfy the HPV chemicals program for this endpoint, a technical discussion will be developed for this endpoint.

Summary: Photodegradation testing and computer modeling will not be conducted for materials in the Petroleum Coke category because they are not subject to this fate process. Instead, a technical discussion on the potential for these materials to photodegrade will be prepared and added to IUCLID, which is the electronic database that will contain data for the HPV chemicals program.

Stability in Water: Hydrolysis of an organic chemical is the transformation process in which a water molecule or hydroxide ion reacts to form a new carbon-oxygen bond. Chemicals that have a potential to hydrolyze include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters (15).

Stability in water can be measured (16) (EPA identifies OECD test guideline 111 as a test method) or estimated (13). An estimation method accepted by the EPA can calculate hydrolysis rate constants for esters, carbamates, epoxides, halomethanes, and selected alkylhalides. Materials in the Petroleum Coke category are not subject to hydrolysis.

To fulfill this endpoint, a technical discussion as to why these materials are not subject to hydrolysis will be developed. The discussion will include a description of the general chemical structure for materials in this category.

Summary: Hydrolysis testing and computer modeling will not be conducted for materials in the Petroleum Coke category because they do not undergo hydrolysis. Instead, a technical discussion on the potential for these materials to hydrolyze will be prepared and added to IUCLID, which is the electronic database that will contain data for the HPV chemicals program.

Chemical Transport and Distribution in the Environment (Fugacity Modeling): Fugacity based multimedia modeling can provide basic information on the relative distribution of chemicals between selected environmental compartments (i.e., air, soil, sediment, suspended solids, water, and biota). The US EPA has agreed that computer modeling techniques are an appropriate approach to estimating chemical partitioning (fugacity is a calculated endpoint and is not measured). A widely used fugacity model is the EQC (Equilibrium Criterion) model (17). EPA cites the use of this model in its document titled *Determining the Adequacy of Existing Data* (18), which was prepared as guidance for the HPV chemicals program.

Because materials in this category are solids whose structure would not be amenable to modeling, a brief technical discussion as to where they would partition in the environment will be developed. The discussion will include a general description of the composition and chemical structure for these materials.

Summary: Fugacity based computer modeling will not be conducted for materials in the Petroleum Coke category because their structure precludes them from being modeled. Instead, a technical discussion on the potential environmental distribution of these materials will be prepared and added to IUCLID, which is the electronic database that will contain data for the HPV chemicals program.

Biodegradation: Biodegradation is the utilization of a chemical by microorganisms as a source of energy and/or carbon. The parent chemical is broken down to simpler, smaller chemicals, which are ultimately converted to an inorganic form such as carbon dioxide, nitrate, sulfate, and water. Assessing the biodegradability of chemicals using a standard testing guideline can provide useful information for evaluating chemical hazard.

Biodegradation can be measured using the OECD test guidelines 301A-F or 302A-C (18). However, because of their structure and physical state, materials in the Petroleum Coke category would not be subject to biodegradative processes that would be measurable with standard testing guidelines. Therefore, a technical discussion will be developed on the potential of these materials to biodegrade.

Summary: Biodegradation testing will not be conducted for materials in the Petroleum Coke category because they do not biodegrade. Instead, a technical discussion on the potential of these materials to degrade will be prepared and added to IUCLID, which is the electronic database that will contain data for the HPV chemicals program.

EVALUATION OF EXISTING ECOTOXICITY DATA AND PROPOSED TESTING

Aquatic Toxicity

There are no data that can be used to evaluate the toxicity of Petroleum Coke to aquatic species. There are three aquatic toxicity endpoints in the HPV chemical program. EPA identifies the following test methods to determine these endpoints: OECD Guideline 203, *Fish Acute Toxicity Test*; Guideline 202, *Daphnia sp., Acute Immobilization Test*; and Guideline 201, *Alga Growth Inhibition Test* (18,19).

The purpose of these aquatic toxicity tests is to determine the concentration or loading of a test material in a test medium that will produce mortality or inhibition to 50% of a population of organisms. Depending on the physicochemical characteristics of the material, it is possible that acute effects may not occur (i.e., a test material may have no to very low water solubility and as a result may not achieve a concentration or loading sufficient to cause effects).

Materials in the Petroleum Coke category are not expected to exhibit measurable water solubility. Therefore, acute aquatic toxicity is not expected. Nevertheless, two of the three aquatic toxicity tests will be conducted to confirm these expectations. Green Coke will be tested using a daphnid and alga species, because it has a higher residual hydrocarbon content. The aquatic invertebrate is generally considered more sensitive to chemical toxicity than fish. If no effects are demonstrated in *Daphnia*, the results will be used as evidence that these materials would not be expected to produce acute effects in fish.

Summary: There are no existing data evaluating aquatic toxicity of petroleum coke. Green Coke will be tested in *Daphnia* immobilization and alga inhibition tests.

Terrestrial Toxicity

Materials in the Petroleum Coke category are sometimes used in a manner that can expose selected terrestrial species to their residues. However, there are no data that can be used to evaluate the potential for toxicity of these materials to terrestrial species. Since these materials can be incorporated in surface soil, their toxicity will be evaluated using test methods described in the OECD Guidelines 207, Earthworm, Acute Toxicity Test, and 208, Terrestrial Plants, Growth Test.

Summary: There are no existing data evaluating the effects of petroleum coke on terrestrial soil species. Green Coke will be tested in earthworm and plant toxicity tests.

Table 2

Matrix of Available Adequate Data and Proposed Testing on Petroleum Coke		
Test	Petroleum Coke (Green) CAS # 64741-79-3	Petroleum Coke (Calcined) CAS # 64743-05-1
Partition Coefficient	N/A	N/A
Water Solubility	N/A	N/A
Biodegradation	N/A	N/A
Environ. Transport	N/A	N/A
Acute Fish	NT	NT
Acute Daphnia	Test	C
Algae	Test	C
Terrestrial	Test	C
Acute Oral	N/A	N/A
Acute Inhalation	NT	NT
Acute Dermal	N/A	N/A
Repeated Dose	Adequate	Adequate
Genotoxicity, in vitro, bacterial	Adequate	C
Genotoxicity, in vitro, non-bacterial	Adequate	C
Genotoxicity, in vivo	Adequate	C
Repro/Developmental	Test	C

Adequate Indicates adequate existing data.

Test Indicates proposed testing

C Indicates category read-across from existing or proposed test data.

N/A Indicates that evaluation of endpoint Not Applicable due to physical-chemical state or route of administration. Technical discussions will be developed to address these endpoints as appropriate.

NT No Testing proposed for reasons provided in test plan.

REFERENCES

Introduction

1. Lee, J. M., Baker, J.J., Murray, D., Llerena, R., and Rolle, J.G. (1997) "Quality analysis of petroleum cokes and coals for export specifications required in use of specialty products and utility fuels." Preprints of Symposia, 214th National Meeting, American Chemical Society Volume 42:844-853.
2. CONCAWE (1993) Petroleum Coke, Product dossier no. 93/105

Health Effects Testing

3. Lipscomb, J., and Lee, S (1982). Health Hazard Evaluation Report No. HETA 81-421-1251, Great Lakes Carbon Corp., Port Arthur, Texas. NIOSH, Cincinnati, Ohio.
4. Acute Toxicity Data, J. Amer. College. Toxicol. , Part B, V. 1 (1990) (15 Suppl 1, S28, 1996).
5. Nau, C.A., Taylor, G.T., and Lawrence, C.H. (1976) Properties and Physiological Effects of Thermal Carbon Black. J. Occup. Med., 18:732-734.
6. Huntingdon Life Sciences (1999). Study No. 97-6119. Calcined coke (F284) & green coke (F-285): fibrogenic screening study in the rat. Report submitted to ARCO 20 October 1999.
7. International research & Development Corp. (1985) Chronic Inhalation toxicity Study of Petroleum Coke (Delayed Process) in Rats and Monkeys. API Publication number 32-30234.
8. Klonne, D. R., Burns, J. M., Halder, C. A., Holdsworth, C. E. and Ulrich, C. E. (1987) Two Year Inhalation Study of Petroleum Coke in Rats and Monkeys. Am. J. Ind. Med., 11:375-389.
9. Wingate, D. A. and Hepler, D. I. (1982) "Carcinogenic Potential of Petroleum Coke and Process Products". Report to American Petroleum Institute on Project 1402 by Elars Bioresearch Laboratories, Inc., API-#PS-22-EBL (131/840).
10. Hazleton Laboratories of America, Inc. (1981) In Vitro and In Vivo Mutagenicity Studies; Delayed Process Coke; (Petroleum coking sample 4-1-140). API Medical Research Publication 28-30728.
11. Hazleton Laboratories of America, Inc. (1981) In Vivo and In Vitro Mutagenicity Studies; Fluid Process Coke; (Petroleum Coking Sample 6-1-468). API Medical Research Publication 28-30726.

Environmental Fate and Effects Testing

12. EPIWIN. (1999) Estimation Program Interface for Windows, version 3.02. Syracuse Research Corporation, Syracuse, NY, USA.
13. US EPA. (1999) The Use of Structure-Activity Relationships (SAR) in the High Production Volume Chemicals Challenge Program. OPPT, EPA.
14. Zepp, R. G., and D. M. Cline. (1977) Rates of Direct Photolysis in the Aqueous Environment. Environ. Sci. Technol. 11:359-366.
15. Atkinson, R. (1989) Kinetics and Mechanisms of the Gasphase Reactions of the Hyroxyl Radical with Organic Compounds. J. Phys. Chem Ref. Data Monograph No. 1. American Institute of Physics and American Chemical Society, New York, NY, USA.
16. Neely, W. B. (1985) Hydrolysis. In: W. B. Neely and G. E. Blau, eds. Environmental Exposure from Chemicals. Vol I., pp. 157-173. CRC Press, Boca Raton, FL, USA.
17. Mackay, D., Di Guardo, A., Paterson, S. and Cowan, C. E. (1996) Evaluating the Environmental Fate of a Variety of Types of Chemicals Using the EQC Model. Environ. Toxicol. Chem. 15:1627-1637.
18. US EPA. (1999) Determining the Adequacy of Existing Data. OPPT, EPA.
19. Organization for Economic Co-operation and Development (OECD). (1999) Draft Guidance Document on Aquatic Toxicity Testing of Difficult Substances. OECD Environmental Health and Safety Publications.

APPENDIX 1

Petroleum Coke Category Constituents by CAS

CAS No.	EINECS No.

64741-79-3 265-080-3

Coke (petroleum)

A solid material resulting from high temperature treatment of petroleum fractions. It consists of carbonaceous material and contains some hydrocarbons having a high carbon-to-hydrogen ratio.

64743-05-1 265-210-9

Coke (petroleum), calcined

A complex combination of carbonaceous material including extremely high molecular weight hydrocarbons obtained as a solid material from the calcining of petroleum coke at temperatures in excess of 1,000^{o}C (1800^{o}F). The hydrocarbons present in calcined coke have a very high carbon-to-hydrogen ratio.

APPENDIX 2

Robust Summaries for Petroleum Coke

Consortium Registration # 1100997

Explanatory Note for Robust Summary Format

The Petroleum HPV Testing Group has elected to use the IUCLID (International Uniform Chemical Information Database) database as the repository for robust summaries for this program. IUCLID has been structured to accommodate a wide variety of data so that it can be used as a repository for all available data on any given chemical or category of chemicals. Many of the data elements (*e.g.* OECD company location and production information, packaging information, emergency procedures, etc.) are outside the SIDS (Screening Information Data Set) requirements of the US HPV Chemical Challenge. Consequently, only those fields relevant to existing data and proposed testing in support of the Petroleum Coke Test Plan are presented in this document.

ROBUST SUMMARY
OF INFORMATION ON

Substance Group # PETROLEUM COKE

Summary prepared by American Petroleum Institute

Creation date: 21-OCTOBER-1999

Printing date: 27 March-2000

Date of last Update: **27 March-2000**

NB. Reliabiliy of data included in this summary has been assessed using the approach described by Klimisch et al.

Klimisch, H. J., Andreae, M. and Tillman, U, (1997)
A systematic approach for evaluating the quality of experimental toxicological and ecotoxicological data.
Regulatory Toxicology and Pharmacology 25, 1-5.

1.1 General Substance Information

Substance type: Petroleum product
Physical status: Solid

Petroleum coke is a black solid produced by the thermal decomposition of petroleum fractions. It may be one of two basic types viz.
GREEN petroleum coke (CAS # 64741-79-3) or
CALCINED petroleum coke (CAS # 64743-05-1).

GREEN coke can be prepared by different processes and as a consequence may be described as either delayed process coke, fluid process coke or flexicoke.

GREEN Coke may contain up to 15% residual hydrocarbon.

CALCINED coke is produced by heating green coke to temperatures up to 1200°C. The resulting material consists essentially of carbon and contains virtually no hydrocarbon.

With one exception, all the studies summarised in this document have been conducted on GREEN coke. It is envisaged that because of the essential difference between GREEN and CALCINED coke (ie a higher hydrocarbon content) the results from the studies on GREEN coke represent a worst case situation and that CALCINED coke would be no more toxic than the GREEN coke samples that have been examined.

1.2 Synonyms

Green coke
Calcined coke
Delayed process coke
Flexicoke
Fluid process coke

2. Physico-chemical data

2.1 Melting Point

Not relevant

2.2 Boiling Point

Not relevant

2.3 Density

Type: Bulk density
Value: 0.7 - 0.95 g/cm^3
Remark: Data summarised by CONCAWE for Green and Calcined coke are as follows:

	Green coke	Calcined coke
Bulk density (kg/dm^3)	0.7 - 0.9	0.75 - 0.95
Real density (kg/dm^3)	1.35 - 1.45	2.06 - 2.16

Reliability: 4, not assignable, - source of original data unknown. However, information may be useful as a guide.

(1)

2.3.1 Granulometry

Not relevant

2.4 Vapour Pressure

Not relevant

2.5 Partition Coefficient

Not relevant

2.6.1 Water Solubility

Insoluble

2.6.2 Surface Tension

Not relevant

2.7 Flash Point

Not relevant

2.8 Auto Flammability

Not relevant

2.9 Flammability

Not relevant

2.11 Oxidizing Properties

Not relevant

3.1.1 Photodegradation

Not relevant

3.1.2 Stability in Water

Petroleum coke is an insoluble solid, consisting essentially of carbon.

3.1.3 Stability in Soil

Stable

3.4 Mode of Degradation in Actual Use

Not relevant

3.5 Biodegradation

Not biodegradable

3.6 BOD5, COD or BOD5/COD Ratio

Not relevant

5.1 Acute Toxicity

5.1.2 Acute Inhalation Toxicity

Species: Rat
Sex: Male/female
Number of Animals: 300
Vehicle: Air as diluent
Exposure time: 6 hour(s)
Value: > 30.7 mg/m^3
Year: 1981
GLP: No
Test substance: Green petroleum coke (CAS # 64741-79-3)
Calcined petroleum coke (CAS # 64743-05-1)
Remark: No studies have been carried out to determine the acute inhalation LC_{50} for petroleum coke following a single exposure. However, longer-term repeat-dose studies have been carried out and a full description of these is given in Section 5.4. In these studies, doses causing no effect after a 6 hour exposure to petroleum coke can be determined; they are as follows:

Species	LC_{50}	Ref.
Rat	>30.7 mg/m^3	IRDC (1985)
Monkey	>30.7 mg/m^3	IRDC (1985)
Rat*	>50.0 mg/m^3	Huntingdon Life Sciences (1999)

* This experiment applies to green and calcined coke.

(5, 6)

5.4 Repeated Dose Toxicity

Species: Rat
Sex: Male/female
Strain: Sprague-Dawley
Route of administration: Inhalation
Exposure period: 2 Years
Frequency of treatment: 6 hours/day, 5 days/week for 2 years (except holidays)
Post. observation. period: None
Doses: 10.2 and 30.7 mg/m^3
Control Group: Yes, concurrent no treatment
LOAEL: 10.7 mg/m^3
Year: 1981
GLP: No

Test substance: Green petroleum coke (Delayed process, micronized), CAS # 64741-79-3.
There was a stable particle size distribution of 3.1 microns over the duration of the study.
A full analysis of the coke sample (approximately 90% carbon) was undertaken at the beginning and at the end of the study. The results showed that there had been no change in the sample. A complete analysis is provided in the report.

Method: Groups of 150 male and 150 female rats underwent whole body exposures to the powdered test materials at concentrations of 10.2 and 30.7 mg/m^3 for 6 hours daily, 5 days a week for 2 years (holidays excepted).
A group of 150 rats of each sex served as untreated controls.

Chamber concentrations were confirmed daily by means of a gravimetric sampling procedure.

Animals were observed twice daily for mortality, weekly for a fuller clinical examination and body weights were also recorded weekly.
10 male and 10 female rats underwent ophthalmologic examination after 3, 6, 12 18 and 24 months of exposure.

Baseline clinical laboratory determinations were made prior to the exposures on 10 rats of each sex from each group. Clinical chemical and haematological evaluations were undertaken on 10 rats of each sex after 1, 3, 6, 12, 18 and 24 months exposure.

All rats dying or sacrificed in extremis were necropsied. Scheduled interim sacrifices were made as follows: 10/sex/group after 5 days and 1 month's exposure; 20/sex/group after 3, 6 and 12 months exposure; 10/sex/group after 18 months exposure. All survivors at 24 months were necropsied. Weights of major organs were recorded at necropsy and a wide range of tissues examined histologically.

A complete cytogenetics evaluation was undertaken on 10 rats of each sex after 5 days and 12 months exposure. Slide preparations were also made for cytogenetic evaluations on 10 rats of each sex after 1, 3 and 6 months exposure. After approximately 22½ months 5-9 rats/sex/group were evaluated. All data were evaluated using appropriate statistical analyses.

Result:

There were no treatment-related effects with repect to clinical condition, growth rates, ophthalmological findings, or serum biochemistry. Furthermore, the cytogenetic evaluations did not differ from those for control animals. Although there were statistically significant differences with respect to segmented neutrophils and lymphocytes between treated and control rats, these were not consistent throughout the study. It was concluded that these changes were probably indicative of a mild inflammatory reaction as a result of deposition of test material in the lungs.

There were significant dose related increases in absolute and relative lung plus trachea weights in the 30.7mg/m^3 group when compared to controls. The effects were noted after 3 months exposure for females and 6 months exposure for males. In the low dose group the effects were seen in females after 18 months and in males after 24 months exposure.

At necropsy it was noted that the exposed rats had gray/black discolouration and foci of the lung and black thoracic lymph nodes at all measurement periods and at termination.

Histological examination showed that chronic pulmonary inflammation had occurred at the 3, 6, 12 and 18 month intervals. Overall, the microscopic changes observed increased in severity with increasing exposure concentration and increasing duration of exposure.
This result is undoubtedly due to deposition of test material and the occurence of an inflammatory response in the lung.
Other than changes in the lung, there were no other significant

	treatment related changes.
Remark:	Note that the results of the cytogenetic evaluations are also reported in section 5.6.
Reliability:	1, valid without restriction. This is a well documented study. All raw data are available for further evaluation if required. Although the study was not conducted according to GLP, there were thorough quality assurance reviews for all segments of the study.

(6)

5. Toxicity

Species:	Rat
Sex:	Male
Strain:	Fischer 344
Route of administration:	Inhalation
Exposure period:	6 hours per day for 5 days
Frequency of treatment:	Daily for 5 days
Post. Observation period:	63 days
Doses:	50 mg/m^3
Control Group:	Yes
NOAEL:	> 50 mg/m^3
Test substance:	Green petroleum coke (CAS # 64741-79-3), 100% pure Calcined petroleum coke (CAS # 64743-05-1), 99.5% pure
Year:	1999
GLP:	Yes
Test condition:	The test materials were delivered as dusts to the 40 litre nose-only inhalation chambers at nominal concentrations of 50 mg/m^3. The control materials titanium dioxide (negative control) and silicon dioxide (positive control) were also delivered as dusts at nominal concentrations of 50 mg/m^3. The actual mass median aerodynamic diameters for the particles of test and control materials was determined and were as follows: Titanium dioxide 0.9433 µm Silicon dioxide 1.737 µm Green coke 2.712 µm Calcined coke 2.692 µm Chamber concentrations were monitored and found to be: Titanium dioxide 53.2 mg/m^3 Silicon dioxide 51.0 mg/m^3 Calcined coke 45.0 mg/m^3 Green coke 58.2 mg/m^3
Method:	Groups of 40 male rats were exposed to either titanium dioxide, silicon dioxide, green coke or calcined coke, each at a nominal concentration of 50 mg/m^3. The nose-only exposures were for 6 hours each day for 5 consecutive days. 10 animals from each group were sacrificed 7, 28 and 63 days after the last exposure. Clinical examination was undertaken throughout the study and body weights were recorded twice pre-test, once during exposure and weekly thereafter. At sacrifice bronchoalveolar lavage was performed and a biochemical and cytological examination was made on the lavage fluid. An extra 10 rats from each group were sacrificed 63 days post exposure. For these animals brain and lung weights were recorded and a complete macroscopic post mortem examination was undertaken. A histopathological examination of the lungs was also made for these extra animals.
Result:	There were no mortalities during the study and there were no significant exposure-related clinical findings, apart from a slight discolouration of the fur of those animals exposed to coke and a slight increase in incidence of chromodacryorrhea in all groups except the TiO_2 group. Apart from marginal weight loss which occured during the exposure period only, no significant effect on growth rates were observed for any treatment group in the study. At the day 7 and day 28 interval, analysis of the bronchiolar lavage fluid did not give any indication of pulmonary toxicity. However, at the day 63 interval a pulmonary effect was noted in

5. Toxicity

the SiO_2 animals and to a lesser degree in the coke exposed animals. The effects in the coke-exposed animals were considered to be a 'slight effect'. The changes noted were: an increase in n-acetylglucosamidase, total protein, total cell count, neutrophil and lymphocyte count.

At 63 days, lung weights of those animals exposed to coke were considered to be comparable with those of both control groups. Although red discolouration of the lungs and parabronchial lymph nodes of the coke-exposed animals was observed this was not regarded as being of toxicological significance. Some inflammation was observed in all treatment groups. The increasing severity of the inflammation was in the order: TiO_2, calcined coke, green coke and SiO_2, the latter being the most severe.

Overall, it was concluded that neither calcined nor green coke caused a fibrogenic effect in the lungs when compared to silicon dioxide and titanium dioxide. Although some pulmonary inflammation occured, it was less severe than that caused by silicon dioxide, and more severe than that caused by titanium dioxide. Inflammation was slightly greater in the green coke when compared to the calcined coke.

Reliability: 1, valid without restriction.

(5)

5. Toxicity

Species:	Primate
Sex:	Male/female
Strain:	Macaca Fascicularis
Route of administration:	Inhalation
Exposure period:	24 Months
Frequency of treatment:	6 hours/day, 5days/week for 24 months (excluding holidays)
Post. observation period:	None
Doses:	10.2 and 30.7 mg/m^3
Control Group:	Yes, concurrent no treatment
Method:	2-year primate inhalation study
Year:	1981
GLP:	No
Test substance:	Green petroleum coke (Delayed process, micronized), CAS # 64741-79-3 There was a stable particle size distribution over the duration of the study of 3.1 microns. A full analysis of the coke sample (approximately 90% carbon) was undertaken at the beginning and at the end of the study. The results showed that there had been no change in the sample over the period of the study. A complete analysis is provided in the report.
Method:	Groups of 4 male and 4 female mature, adult monkeys underwent whole body exposures to the powdered test materials at concentrations of 10.2 and 30.7 mg/m^3 for 6 hours daily, 5 days a week for 2 years (holidays excepted). A group of 4 monkeys of each sex served as untreated controls. Chamber concentrations were confirmed daily by means of a gravimetric sampling procedure. Animals were observed twice daily for mortality, monthly for a fuller clinical examination and body weights were also recorded monthly. Ophthalmologic examinations were conducted on all monkeys prior to exposure and after 1, 3, 6, 12 18 and 24 months of exposure. Baseline clinical laboratory determinations were made twice prior to the exposures on all monkeys. Clinical chemical and haematological evaluations were undertaken on all monkeys after 1, 3, 6, 12, 18 and 24 months exposure. All survivors at 24 months were necropsied. Weights of major organs were recorded at necropsy and a wide range of tissues examined histologically. After 24 months exposure all monkeys were evaluated for calcium and phosphorus levels in bone ash, using samples from femur and rib. All data were evaluated using appropriate statistical analyses.
Result:	There were no treatment related effects with repect to clinical condition, growth rates, ophthalmological findings, serum biochemistry or haematological parameters. There were significant dose related increases in absolute and relative lung plus trachea weights in both dose groups of male and females after 24 months exposure. At necropsy it was noted that the exposed monkeys had gray/black discolouration and foci of the lung and black thoracic lymph nodes.

Histological examination showed trace to moderate

accumulations of alveolar macrophages containing test material in both sexes and at both dose levels. Similar accumulations of macrophages were also seen in the thoracic lymph nodes and in paratracheal lymphoid tissue. There were no other significant treatment related changes.

Reliability: 1, valid without restriction. This is a well documented study. All raw data are available for further evaluation if required. Although the study was not conducted according to GLP, there were thorough quality assurance reviews for all segments of the study.

(6, 7)

5.5 Genetic Toxicity 'in Vitro'

Type: Ames test
System of testing: Assays carried out using *S. typhimurium* strains TA 1535, TA 1537, TA 1538, TA 98 and TA 100
Concentration: 5 concentrations: up to 10,000µg/0.1ml
Metabolic activation: With and without
Result: Negative
Method: Salmonella typhimurium Reverse Mutation Assay
Year: 1979
GLP: No data
Test substance: Green petroleum coke (Delayed process, micronized)
CAS # 64741-79-3
Sample 4-1-140.
Black powder.
An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069)
Solvent used: DMSO
Concentrations for toxicity testing:
1,000; 100; 10; 1.0 and 0.1µg/ml
Concentrations for mutagenicity testing were:
123.5; 370.4; 1111.1; 3333.3 and 10,000µg/0.1ml

Method: An S9 mammalian liver cell fraction was obtained from Sprague Dawley rats that had been induced with Aroclor® 1254 at a dose of 500mg/Kg i.p. for 5 days. 0.5ml of the S9 fraction was used in the Ames assays. The assay was also run using a negative, solvent (DMSO) control and a positive control.

Result: Although the highest concentration tested (10,000µg/0.1 ml) did not show bacterial growth inhibition, the limits of solubility of the test material had clearly been reached. Using the 5 Salmonella typhimurium strains, the delayed process coke sample was not mutagenic at any of the concentrations tested either with or without metabolic activation.

Reliability: 1, valid without restriction. Although this study was probably not conducted to GLP the test parameters were based on a well established procedure and was conducted by a well established laboratory. The report describes fully the procedures used and the results obtained.

(2, 3)

5. Toxicity

Type:	Ames test
System of testing:	Assays carried out using S. typhimurium strains TA 1535, TA 1537, TA 1538, TA 98 and TA 100
Concentration:	5 concentrations: up to 10,000µg/0.1ml
Metabolic activation:	With and without
Result:	Negative
Method:	Salmonella typhimurium Reverse Mutation Assay
Year:	1979
GLP:	No data
Test substance:	Green coke (Fluid process) CAS # 64741-79-3 Sample 6-1-468. Black powder. An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069) Solvent used: DMSO Concentrations for toxicity testing: 1,000; 100; 10; 1.0 and 0.1µg/ml Concentrations for mutagenicity testing were: 123.5; 370.4; 1111.1; 3333.3 and 10,000µg/0.1ml.
Method:	An S9 mammalian liver cell fraction was obtained from Sprague Dawley rats that had been induced with Aroclor® 1254 at a dose of 500mg/Kg i.p. for 5 days. 0.5ml of the S9 fraction was used in the Ames assays. The assay was also run using a negative, solvent (DMSO) control and a positive control.
Result:	Heavy chemical precipitation occured at the three highest concentrations tested which meant manual counting of the number of revertant colonies at these levels. At the highest concentration (10,000µg/0.1ml) the revertant colonies of TA1538, TA98 and TA100 could not be counted due to a heavy bacterial contamination. However, no mutagenic activity was found at any of the other concentrations tested for any of the Salmonella strains either with or without metabolic activation. In conclusion, the sample of fluid process coke was not mutagenic in the assay.
Reliability:	2, valid with restrictions. Although the study was partially compromised due to bacterial contamination, it was nevertheless reported fully and raw data are available in the report for evaluation. The results are consistent with those from other studies on similar materials.

(2, 4)

5. Toxicity

Type:	Mouse lymphoma assay
System of testing:	Assay carried out using L5417Y mouse lymphoma cell line
Concentration:	8 concentrations up to 2000 µg/ml
Metabolic activation:	With and without
Result:	Negative
Method:	Mouse lymphoma assay
Year:	1979
GLP:	No data
Test substance:	Green coke (Delayed process) CAS # 64741-79-3 Sample 4-1-140. Black powder. An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069). Solvent used: DMSO Concentrations for toxicity testing: 5; 1,000; 10,000; 50,000 and 100,000 µg/ml Concentrations for mutagenicity testing were: 600; 800; 1,000; 1,200; 1,400; 1,600; 1,800 and 2,000 µg/ml Positive control substance: ethyl methane sulphonate not requiring activation, Promitogen requiring S 9 activation.
Method:	The study was conducted according to guideline. S-9 cofactor was obtained from Sprague Dawley rats that had been induced with PCBs.
Result:	No toxicity to mouse lymphoma cells was observed at concentrations up to 2000µg/ml. Insolubility of test compound precluded testing at higher concentrations. The values for spontaneous and induced mutation frequencies were within acceptable limits. The petroleum coke sample 4-1-140 did not induce forward mutations at the thymidine kinase (TK) locus in L5178Y, clone 3.7.2, Mouse Lymphoma cells either with or without metabolic activation.
Reliability:	1, valid without restriction.

(2, 3)

5. Toxicity

Type:	Mouse lymphoma assay
System of testing:	Assay carried out using L5417Y mouse lymphoma cell line
Concentration:	8 concentrations up to 2,000 µg/ml
Metabolic activation:	With and without
Result:	Negative
Method:	Mouse lymphoma assay
Year:	1979
GLP:	No data
Test substance:	Green coke (Fluid process) CAS # 64741-79-3 Sample 6-1-468. Black powder. An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069). Solvent used: DMSO Concentrations for toxicity testing: 5; 1,000; 10,000; 50,000 and 100,000 µg/ml Concentrations for mutagenicity testing were: 600; 800; 1,000; 1,200; 1,400; 1,600; 1,800 and 2,000 µg/ml Positive control substance: ethyl methane sulphonate not requiring activation, Promitogen requiring S 9 activation.
Method:	The study was conducted according to guideline. S 9 cofactor was obtained from Sprague Dawley rats that had been induced with PCBs.
Result:	No toxicity to mouse lymphoma cells was observed at concentrations up to 2000µg/ml. Insolubility of test compound precluded testing at higher concentrations. The muation frequencies for the positive controls were within acceptable limits. The spontaneous mutation frequency (2.6) for the solvent controls without metabolic activation was lower than generally seen for DMSO. However, this lower frequency was attributed to an unusually high cell count for the non-selective count and it was concluded that this had probably been caused by a dilution error. The spontaneous background mutational frequency for the media (5.5) was comparable to previous results obtained in the laboratory. Therefore, the media control mutation frequency (5.5) rather than that for the solvent (2.6) was used to evaluate the non-activated portion of the assay. This approach is further supported by the fact that there were no significant increases in the number of TK-/- mutant colonies on the selective medium plates from the test doses as compared to the solvent control. In conclusion, the petroleum coke sample 6-1-468 did not demonstrate a positive response and is not mutgenic in the L5178Y Mouse Lymphoma assay, either with or without metabolic activation.
Reliability:	2, valid with restrictions. A posible dilution error may have compromised the results for the negative controls. However, the overall result of the assay is in-line with similar assays on similar materials. Furthermore the error gives conclusions that are more conservative than would be otherwise expected had the negative control values been greater.

(2, 4)

5.6 Genetic Toxicity 'in Vivo'

Type:	2 year inhalation study
Species:	Rat
Sex:	Male/female
Strain:	Sprague-Dawley
Route of administration:	Inhalation
Exposure period:	Up to 2 years
Doses:	10.2 and 30.7 mg/m^3
Result:	Negative
Year:	1981
GLP:	Yes
Test substance:	Green petroleum coke (Delayed process, micronized). CAS # 64741-79-3 There was a stable particle size distribution of 3.1 microns over the duration of the study. A full analysis of the coke sample (approximately 90% carbon) was undertaken at the beginning and at the end of the study. The results showed that there had been no change in the sample. A complete analysis is provided in the report.
Method:	Groups of 150 male and 150 female rats underwent whole body exposures to the powdered test materials at concentrations of 10.2 and 30.7 mg/m^3 for 6 hours daily, 5 days a week for 2 years (holidays excepted). A group of 150 rats of each sex served as untreated controls. Chamber concentrations were confirmed daily by means of a gravimetric sampling procedure. Animals were observed twice daily for mortality, weekly for a fuller clinical examination and body weights were also recorded weekly. A complete cytogenetics evaluation was undertaken on 10 rats of each sex after 5 days and 12 months exposure. Slide preparations were also made for cytogenetic evaluations on 10 rats of each sex after 1, 3 and 6 months exposure. After approximately 22½ months 5-9 rats/sex/group were evaluated. All data were evaluated using appropriate statistical analyses.
Remark:	This study was made on an extra group of rats that had been included in a two year inhalation study with green coke. (See section 5.4.).
Result:	Although there were statistically significant differences with respect to segmented neutrophils and lymphocytes between treated and control rats, these were not consistent throughout the study. It was concluded that these changes were probably indicative of a mild inflammatory reaction as a result of deposition of test material in the lungs. Overall, the cytogenetic evaluations did not differ from those for control animals.
Reliability:	1, valid without restriction.

(6)

5. Toxicity

Type:	Cytogenetic assay
Species:	Rat
Sex:	Male
Strain:	Sprague-Dawley
Route of administration:	Inhalation
Exposure period:	28 days
Doses:	0, 12.45 and 45.34 µg/l
Year:	1979
GLP:	No data
Test substance:	Green coke (Delayed process) CAS # 64741-79-3 Sample 4-1-100. Black powder. An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069). Dust atmosphere generated using a cascade impactor to achieve particles < 5µ.
Method:	Groups of 8 sexually mature male Sprague Dawley rats were exposed to powdered coke sample at the following nominal concentrations: 0, 10 and 40µg/l. The low dose animals were exposed 6 hours each day, 5 days per week for 20 exposures. The high dose animals were exposed 6 hours each day for 5 days only. Controls were held for the same time as the low dose group animals. The day after each animal's last exposure colchicine was administered to inhibit mitosis. Two hours later the animals were sacrificed and bone marrow smears were made from the femur. The slides were photographed for a permanent record.
Result:	Gravimetric samples collected throughout the study established the actual exposure concentrations to be 0, 12.45 and 45.34 µg/l. Appearance, behaviour and growth rates were unaffected by exposure. There was some deposition of test material on the fur of the animals exposed to the coke sample. There were no significant differences among any of the groups when comparing the percentages of cells containing chromosome breaks or severely damaged cells. There was however a significant increase in the number of chromatid breaks, markers and total aberrations in the highest dose group when compared to controls. Mitotic indices were unaffected by exposure to petroleum coke. In conclusion, petroleum coke sample 4-1-100 produced structural mutations in bone marrow cells of rats exposed to 45.34 µg/l by inhalation for 5 consecutive days. No effects were noted at the lower dose level tested over a period of 20 exposures.
Reliability:	3, invalid. The microscope slides had been misread in this study, thus invalidating the findings.

(3)

5. Toxicity

Type:	Cytogenetic assay
Species:	Rat
Sex:	Male
Strain:	Sprague-Dawley
Route of administration:	Inhalation
Exposure period:	28 days
Doses:	0, 10 and 40 µg/l
Result:	Negative
Year:	1979
GLP:	No data
Test substance:	Green coke (Fluid process) CAS # 64741-79-3 Sample 6-1-648. Black powder. An analysis of the sample has been conducted and reported elsewhere (Gulf R&D Co. Report No. 553RL069). Test atmosphere generated using a wright dust feed mechanism. The atmospheres consisted of particles < 5µ. Test atmosphere concentrations were 0, 10 and 40 µg/l.
Method:	Groups of 8 sexually mature male Sprague Dawley rats were exposed to the powdered coke sample at the following nominal concentrations: 0, 10 and 40µg/l. The low dose animals were exposed 6 hours each day, 5 days per week for 20 exposures. The high dose animals were exposed 6 hours each day for 5 days only. Controls were held for the same time as the low dose group animals. The day after each animal's last exposure colchicine was administered to inhibit mitosis. Two hours later the animals were sacrificed and bone marrow smears were made from the femur. The slides were photographed for a permanent record.
Result:	Appearance, behaviour and growth rates were unaffected by exposure to petroleum coke. There were no statistically significant differences between test groups and controls for any of the parameters measured. Sample 6-1-468 did not cause any cytogentic effects in male rats exposed at levels up to and including 40 µg/l.
Reliability:	1, valid without restriction.

(4)

5.7 Carcinogenicity

Species:	Mouse
Sex:	Male/female
Strain:	C3H
Route of administration:	Dermal
Exposure period:	Lifetime
Frequency of treatment:	3 times weekly
Post. Observation period:	None
Doses:	Coke samples at 25%, condensate sample at 100%
Result:	Negative
Control Group:	Yes
Method:	Repeated dose dermal carcinogenicity study
Year:	1979
GLP:	Yes
Test substance:	Green petroleum coke CAS # 64741-79-3 Calcined petroleum coke CAS # 64743-05-1 The following samples were tested: Sample No. 3-1-134 Green coke (Ground, solid condensed emission/delayed process coke), tested as a 25% suspension in mineral oil. Sample No. 4-1-140 Green coke (Delayed process, micronized), tested as a 25% suspension in mineral oil. Sample No. 6-1-468 Green coke (Fluid process, micronized), tested as a 25% suspension in mineral oil. Sample No. 7-1-100 Process water from delayed process coke, tested undiluted. Positive control substance Benzo-a-pyrene, tested as 0.05% and 0.15% solutions in mineral oil Vehicle control Veterinary grade mineral oil
Method:	The 4 test materials and the vehicle control were applied (100 µl) three times weekly to the shaven dorsal surface of groups of 25 male and 25 female mice for their lifetimes. Treatment was stopped and the animals sacrificed when the study director considered it to be appropriate for humane reasons. The benzo-a-pyrene positive control groups were treated twice weekly for their lifetimes. Additionally 25 male and 25 female control mice were included. These animals were shaved only, without further treatment. The animals, aged 66 days at the commencement of the study, were checked daily for viability and three times weekly for a more comprehensive clinical examination. Body weights were recorded every two weeks. Pathology was carried out on all mice that died and histology was conducted on a wide range of tissues and organs.

5. Toxicity

Result: The positive control groups had decreased survival times compared with the vehicle and "shaved only" negative controls. Survival was unaffected by treatment with any of the coke

samples or the condensate.
Although some body weight effects were recorded, there were no consistent treatment-related trends for the coke and condensate samples.
No neoplatic changes were recorded at the treatment site for the negative controls or any of the coke and condensate test groups. In contrast, squamous cell neoplasms developed at the treatment sites in the positive control animals.
The incidence of acanthosis and hyperkeratosis observed together with the number of spontaneous mammary tumours recorded in the animals is shown in the table below:

Sample	Acanthosis (%)		Hyperkeratosis (%)		No. of mice with mammary tumours
	M	F	M	F	
Shaved	21	16	16	12	6
Vehicle	42	46			4
3-1-134	68	83	12	9	4
4-1-140	79	92	38	42	9
6-1-468	96	72	71	36	8
7-1-100	12	17	48	54	5

In conclusion, the study demonstrated that neither the coke samples nor the condensate were carcinogenic in a lifetime skin painting study in mice.
Apart from an increased incidence of acanthosis and hyperkeratosis of the skin at the treatment site, there were no other treatment related effects.

Reliability: 1, valid without restriction. This is a well described and documented study. All the data are presented such that independent evaluation could be undertaken if required.

(9)

5.11 Experience with Human Exposure

Remark: Although the effects of green or clacined coke on man have not been studied, there have been several epidemiology studies conducted at manufacturing plants where petroleum coke was in use. The common feature of these studies was the examination of the effects of dusts and PAHs on the workforce, but in none of them was it possible to identify the contribution of coke to the effects observed.

One study was conducted to evaluate respiratory function and reported respiratory disease among workers exposed to petroleum coke dust. In this study, 90 employees (55% of the workforce) participated in a medical investigation which included a respiratory questionnaire, pulmonary function tests (PFT) and chest X-ray. The medical evaluation revealed abnormal PFT results among 9 (10%) current employees. The PFT abnormalities were significantly related to dust exposure as

measured by length of employment, age and a history of working for 5 years or longer in the mobile equipment department. Chest

X-rays showed no evidence of pneumoconiosis. Although no pneumoconiosis was detected, the medical study did find evidence of occupationally-related pulmonary function abnormalities.

(8)

6 References

1 CONCAWE (1993)
Petroleum Coke.
Product dossier No. 93/105
CONCAWE, Brussels

2. Gulf Research & Development Co. (1980)
American Petroleum Institute Project PS-22-GRD(840). Comprehensive analysis of Petroleum Coke Products.
Report No. 553RL069.

3 Hazleton Laboratories of America, Inc. (1981)
In Vitro and In Vivo mutagenicity studies; Delayed process coke; (Petroleum coking sample 4-1-140)
API Medical Research Publication 28-30728, January 20, 1981.

4 Hazleton Laboratories of America, Inc., (1981)
In Vivo and In Vitro mutagenicity studies; Fluid process coke; (Petroleum coking sample 6-1-468)
API Medical Research Publication 28-30726, January 20, 1981.

5 Huntingdon Life Sciences (1999)
Study No. 97-6119. Calcined coke (F-284) & Green coke (F-285): Fibrogenic screening study in the rat
Report submitted to ARCO 20 October 1999

6 International Research and Development Corporation (1985)
Chronic Inhalation toxicity study of petroleum coke (delayed process) in rats and monkeys.
Report prepared for the American Petroleum Institute.
API Publication number 32-30234.

7 Klonne, D.R., Burns, J.M., Halder, C.A., Holdsworth, C.E. and Ulrich, C.E. (1987)
Two year Inhalation Study of Petroleum Coke in Rats and Monkeys.
American Journal of Industrial Medicine, Vol 11. pp. 375-389

8 Lipscomb, J and Lee, S. (1981)
Health hazard evaluation report No. HETA 81-421-1251, Great lakes Carbon Corp., Port Arthur, Texas
NIOSH, Cincinnati, Ohio

9 Wingate, D.A. and Hepler,D.I., (1982)
Carcinogenic potential of petroleum coke and process products API-#PS-22-EBL (131/840)
Report to American Petroleum Institute on Project 1402 by Elars Bioresearch Laboratories,Inc.